

RECEIVED  
SUPREME COURT  
STATE OF WASHINGTON

2009 MAR 13 P 2:56

BY RONALD R. CARPENTER

---

Supreme Court Case No. 82264-6

CLERK *h*

---

**WASHINGTON STATE SUPREME COURT**

---

Julie Anderson, individually and on behalf of Dalton Anderson, a minor, and  
Darwin Anderson,

Appellant,

vs.

Akzo Nobel Coatings, Inc. and Keith Crockett,

Respondents

---

**APPELLANTS' REPLY BRIEF**

---

John R. Connelly, Jr, WSBA No. 12183  
Lincoln C. Beauregard, WSBA No. 32878  
CONNELLY LAW OFFICES  
2301 N. 30<sup>th</sup> Street  
Tacoma, Washington 98403  
(253) 593-5100  
Fax (253) 593-0380

 **ORIGINAL**

## TABLE OF CONTENTS

I.	REPLY .....	1
IV.	ARGUMENT .....	1
A.	With respect to comparative fault, the Anderson family raised the same issues before the trial court, and the only evidence produced by Akzo Nobel concerned the manner in which Ms. Anderson carried out the essential functions of her job during pregnancy. ....	1
B.	The proper application of the <i>Frye</i> test is unclear, and, for assorted reasons, the legal ambiguity should be remedied by embracing the “methodology” based application or jettisoning the <i>Frye</i> test altogether.....	2
C.	The provisions set forth under RCW 49.17.160 do not provide an “adequate” legal mechanism upon which to obtain relief for a retaliatory discharge. ....	27
V.	CONCLUSION.....	33

## TABLE OF AUTHORITIES

### CASES

<i>Berry v. CSX Transportation</i> , 709 So. 2d 552 (1998).....	3
<i>Callahan v. Cardinal Glennon Hosp.</i> , 863 S.W. 2d 852 (Mo. 1983) .....	15
<i>Christophersen v. Allied-Signal Corp.</i> , 902 F.2d 362, 366 (5 <sup>th</sup> Cir. 1990).....	13
<i>Donaldson v. Central Illinois Public Service Co.</i> , 199 Ill.2d 63, 77-79, 262 Ill. Dec. 854, 767 N.E.2d 314 (2002).....	22
<i>Donovan v. OSHRC</i> , 713 F.2d 918 (2 <sup>nd</sup> Cir. 1983).....	30
<i>Earl v. Cryovac, A Div. of W.R. Grace Co.</i> , 115 Idaho 1087, 1095, 772 P.2d 725 (1989).....	15
<i>Ferebee v. Chevron Chemical Co.</i> , 736 F.2d 1529, 1536 (D.C. Cir. 1984) .....	15
<i>Grady v. Frito-Lay, Inc.</i> , 576 Pa. 546, 558-61, 839 A.2d 1038 (2003).....	22
<i>Grant v. Boccia</i> , 133 Wn. App. 176, 137 P.3d 20 (2006).....	22
<i>Heller v. Shaw Industries, Inc.</i> , 167 F.3d 146 (3 <sup>rd</sup> Cir. 1999).....	14
<i>In re Commitment of Simons</i> , 213 Ill.2d 523, 290 Ill.Dec. 610, 821 N.E.2d 1184 (2004).....	22
<i>Kennedy v. Collagen Corp.</i> , 161 F.3d 1226, 1229 (9 <sup>th</sup> Cir. 1998).....	14
<i>Korslund v. Dyncorp Tri-Cities Services, Inc.</i> , 156 Wn.2d 168, 125 P.3d 119 (2005).....	27
<i>McCulloch v. H.B. Fuller Co.</i> , 61 F.3d 1038 (2 <sup>nd</sup> Cir. 1995).....	15
<i>State v. Baby</i> , 404 Md. 220, 946 A.2d 463 (2008).....	23
<i>State v. Cauthron</i> , 120 Wash. 2d 979, 887, 846 P.2d 502 (1993).....	13, 24
<i>Wilson v. The City of Monroe</i> , 88 Wn. App. 113, 943 P.2d 1134 (1997).....	32

**STATUTES**

RCW 49.17.160 .....passim

## I. REPLY

The Anderson family submits this reply to Akzo Nobel's responsive brief. Akzo Nobel's arguments and conclusions with respect to the application of the *Frye* test are flawed, and the trial court erred as an extension thereon. With respect to the *Frye* test, the law does not and should not require scrutiny of the data underlying epidemiological studies and many courts have rejected that approach. In addition to committing error with respect to the *Frye* test, the trial court also erred with regard to the issues of comparative fault and retaliatory discharge. As is set forth herein, this matter should be reversed and remanded for a proper adjudication on the merits.

## IV. ARGUMENT

- A. **With respect to comparative fault, the Anderson family raised the same issues before the trial court, and the only evidence produced by Akzo Nobel concerned the manner in which Ms. Anderson carried out the essential functions of her job during pregnancy.**

On the issue of comparative fault, Akzo Nobel now claims that it did not argue that Ms. Anderson could be held comparatively at fault for performing the essential functions of her job. In the response brief, Akzo Nobel recasts the issue and claims that, instead, on the issue of comparative fault, Akzo Nobel presented evidence supportive of the notion that "*if Ms. Anderson mixed paint while she was pregnant, it was directly contradictory to the directions and admonitions she was receiving*

from her co-corkers.”<sup>1</sup> Even accepting, without agreeing, that Akzo Nobel’s characterization of the issue is correct, the primary issue which was raised by the Anderson family still remains: can Ms. Anderson be held comparatively at fault for Dalton’s injuries for performing the essential functions (paint mixing) of her job while pregnant? As a matter of public policy, for the reasons set forth in the opening brief, the Anderson family submits that Akzo Nobel failed to come forward with evidence supporting the claim that Ms. Anderson is comparatively at fault for Dalton’s injuries. The only evidence which was presented by Akzo Nobel was that Ms. Anderson performed the essential functions of her job.

**B. The proper application of the *Frye* test is unclear, and, for assorted reasons, the legal ambiguity should be remedied by embracing the “methodology” based application or jettisoning the *Frye* test altogether.**

The Anderson family’s causation theory is that organic solvent exposure in the workplace cause fetal brain malformation/encephalopathy.<sup>2</sup> On this, all of the experts for the Anderson family and Akzo Nobel agree.<sup>3</sup> Akzo Nobel urges that *Frye* requires pinpointing a more specific diagnosis and offered up its own, PMG, for purposes of the *Frye* test in this matter. The Anderson family does not agree that Dalton suffers from PMG, and neither do any of the

---

<sup>1</sup> Akzo Nobel Response Brief, Page 19.

<sup>2</sup> Because the law is unclear as to the proper manner in which to identify the causation theory at issue, it should be noted that the Anderson family’s causation theory could also be framed that *in utero* organic solvent exposure causes cognitive delays such as those suffered by Dalton.

<sup>3</sup> CP 209-15 (Exhibit 21 to Declaration of Beauregard (Koren Deposition Page 16))

treating physicians. To the extent a specific name of the malformation is required under the *Frye* test, the Anderson family relies upon the unbiased treating physicians who have diagnosed Dalton with a neuronal migration disorder.<sup>4</sup>

The Florida Supreme Court already held that it is generally accepted in the scientific community that organic solvent exposure in the workplace causes brain damage/encephalopathy. See *Berry v. CSX Transportation*, 709 So. 2d 552 (1998). The *Berry* Court did not endeavor to determine that each of the specific subgroups of encephalopathy was *Frye* tested and decided that that level of hyper-technical argument was the point at which the issue of causation became that for the jury. *Id.* And courts all over the country have declined to analyze similar issues beyond that the more general “encephalopathy” diagnosis when evaluating the underlying scientific veracity of the principles involved.<sup>5</sup>

Contrary to Akzo Nobel’s representations, the Anderson family’s causation theory has always been framed generally, with respect to brain malformations/encephalopathy, and the proceedings during the subsequent

---

<sup>4</sup> Exhibit 24 to Declaration of Beauregard (Deposition of Glass Page 64 to 65).

<sup>5</sup> “...encephalopathy occurs when there has been an alteration to the brain and central nervous system function due to exposure to various toxins. See generally Neil L. Rosenberg, M.D., *Occupational and Environmental Neurology*, 116-17 (1995) (herein *Occupational and Environmental Neurology*). As explained in William N. Rom, M.D. (ed.) *Environmental and Occupational Medicine* at 849 (1992): The nonspecific effects of long-term exposure to solvents range from a general negative affective state to a subtle reduction in functional reserve capacity to perform well when fatigued or in a distracting environment, to mild slowing of psycho-motor performance, to memory disturbance, and finally to severe intellectual deficits. The most severe condition, which has been called psycho-organic syndrome, presenile dementia, and severe chronic toxic encephalopathy, is also the most controversial. Although the existence of chronic solvent encephalopathy

motion for reconsideration referenced by Akzo Nobel were an attempt to clarify that precise mischaracterization of the issue on the part of the trial court. For example, on page 3 of in the Anderson family's first responsive brief to Akzo Nobel's *Frye* motion, the Anderson family argued that *Berry* was more or less determinative, and, therefore, the *Frye* test was arguably "not implicated" premised upon the conclusions reached in *Berry*.<sup>6</sup> The Anderson family now maintains this causation theory and reliance upon *Berry* herein.<sup>7</sup>

Akzo Nobel tries to distinguish *Berry* by purporting, on page 33 of the response brief, that "*Findings of fact in unrelated cases are hearsay, and not admissible in evidence.*" This is a disturbingly misleading assertion on the part of Akzo Nobel in that, at the *Frye* test/motion in *limine* phase, the relevance of *Berry* is to demonstrate that another supreme court has applied the *Frye* test under very similar circumstances and it is well established and expressly stated by this Court that the opinions from other courts are to be considered: "Thus, we examine the record, available literature of law reviews and other journals, and the **cases of other jurisdictions.**" *Cauthron*, 120 Wash. 2d. at 888 (emphasis added). The *Cauthron* Court also noted that the importance and utility of *Frye* precedent explaining that "Once this court has made a determination

---

has been questioned, experts now generally agree that it occurs but not on its prevalence." *Berry*, 709 So. 2d at fn 2 (1998).

<sup>6</sup> CP 547-76

<sup>7</sup> *Id.*



that the *Frye* test is met as to a specific novel scientific theory or principle, the trial courts can generally rely upon that determination as settling such theory's admissibility in future cases." *Id.* at 506 fn3. Certainly the related precedent of other supreme courts should be given great weight in this context. *Id.*

*Berry* involved organic solvent exposure to *adults* in the workplace versus the *unborn children* of mothers in the workplace, as is the case here. However, it is not disputed by the experts that organic solvents "*are fat soluble, they go right through the placenta, dissolve right into the amniotic fluids inside of the uterus, and they've been found in the cell membranes of fetuses.*"<sup>8</sup> In that regard, when deposed, Akzo Nobel's expert, Dr. Koren explained:

Q. Sure. How about this, would you agree with this statement: "Symptomatic exposure appears to confer an unacceptable level of fetal exposure and should be avoided by appropriate protection and ventilation"?

A. I agree.<sup>9</sup>

And Dr. Koren also agreed as follows:

Q. The adult may be okay but the unborn's brain, which is much more sensitive, it is still developing, the baby needs its own guidelines?

A. That's correct.<sup>10</sup>

---

<sup>8</sup> CP 577-768 (Exhibit 23 to Declaration of Beauregard (Schultz Deposition Page 65 lines 20 to 25 to Page 26 lines 1 to 4))

<sup>9</sup> CP 577-768 (Deposition of Koren)

<sup>10</sup> *Id.*

It is very much in accord with Washington law to follow precedent such as *Berry* when conducting a *Frye* test, and *Berry* is nearly conclusive as to the *Frye* issues involved in this case. Additionally, with respect to medical causation, experts for the Anderson family, such as Dr. Khattak, and experts for Akzo Nobel, such as Dr. Koren, are in agreement that organic solvent exposure in the workplace causes fetal brain malformations. When deposed, Dr. Koren explained:

Q. So you've written articles supporting the premise that organic solvent exposure to pregnant women causes -- affects brain development in fetuses; is that right?

A. Yes.<sup>11</sup>

To the extent that "brain malformations" are defined generally, rather than specifically by type of malformation as discerned by the shape as it appears on an MRI image, the experts are in absolute agreement, organic solvent exposure in the workplace causes fetal brain malformations.

1. **Akzo Nobel attempts to capitalize on the *Frye* test by hiring out of state experts to render extremely nuanced diagnosis about Dalton's condition in order to try and erroneously reframe and confuse the issues:**

Akzo Nobel's responsive arguments and tactics highlight the many problems with the *Frye* test. For example, throughout the course of these proceedings, Akzo Nobel has tried to assign a new name to the malformation of Dalton's brain called "polymicrogyria" or "PMG" and then explain why the *Frye* test is not satisfied with respect the specific brain malformation which was unilaterally selected by Akzo Nobel. In

that respect, the Anderson family is *not* contending and does *not* agree that Dalton suffers from PMG, the name of the malformation assigned by Akzo Nobel for the first time ever during litigation. Moreover, at no point in time have any of Dalton's treating physicians diagnosed Dalton with PMG. All of the references in the record to PMG are red herrings which were tactically created by Akzo Nobel in order to capitalize on the availability, and unpredictability of *Frye* as applied in Washington. Akzo Nobel is trying to force the Anderson family to prove a causation theory with respect to PMG when the Anderson family, and the treating physicians, do not agree that Dalton suffers from PMG.

By contrast, the Anderson family *is* claiming that Dalton suffers from a brain malformation, otherwise referred to as encephalopathy, and can be specifically referred to as a neuronal migration defect.<sup>12</sup> All of the treating physicians and hired experts (plaintiffs and defense) agree that Dalton suffers from a brain malformation.<sup>13</sup> All of the treating physicians and hired experts agree that Dalton suffers from encephalopathy.<sup>14</sup> The treating physicians and experts retained by the Anderson family agree that the subgroup of brain malformation/encephalopathy suffered by Dalton is referred to as a neuronal migration disorder.<sup>15</sup> The Anderson family's

---

<sup>11</sup> CP 209-15 (Exhibit 21 to Declaration of Beauregard (Koren Deposition Page 16))

<sup>12</sup> CP 547-76

<sup>13</sup> Exhibit 24 to Declaration of Beauregard (Deposition of Glass Page 64 to 65).

<sup>14</sup> *Id.*

<sup>15</sup> *Id.*

causation theory is, and always has been, that Dalton's brain malformations (encephalopathy) were caused by *in utero* organic solvent exposure during the timeframe that Ms. Anderson worked at Akzo Nobel.<sup>16</sup>

Because it became clear during discovery that the experts for the Anderson family and Akzo Nobel were in agreement, and that it is generally accepted in the medical community that *in utero* organic solvent exposure in workplace causes fetal brain malformations and/or encephalopathy, in the moving papers below, Akzo Nobel argued that the Anderson family could not prove that the *specific type of brain malformation* had been proven to be caused by organic solvent exposure. Thereafter, the parade of diagnostic arguments ensued with Akzo Nobel opting to assign whichever specific type of brain malformation, *e.g.* PMG, was statistically least supported by the existing epidemiological research.<sup>17</sup>

In response to Akzo Nobel's arguments, the Anderson family explained to the trial court that the experts were in agreement: organic solvent exposure in the workplace causes fetal brain malformations.<sup>18</sup>

---

<sup>16</sup> CP 547-76

<sup>17</sup> For example, on page 14 of the responsive brief, Akzo Nobel contends that "There is no support in the medical or scientific literature for the theory of causation linking PMG and exposure to organic solvents." Again, the Anderson family is not trying to prove that PMG and organic solvents have been linked. The Anderson family does not even agree that Dalton suffers from PMG. Dalton's treating physicians do not agree that Dalton suffers from PMG. Akzo Nobel's briefing is littered with references to this "PMG" condition simply to confuse the issues, and to also try and confuse this Court.

<sup>18</sup> CP 547-76

Then, additionally, to support the premise of causation with respect to the more specific diagnosis in relation to Dalton's neuronal migration defect, the Anderson family cited the 1999 JAMA article.<sup>19</sup> In that article, which was co-authored by Dr. Khattak and Dr. Koren, it was determined that pregnant mothers who were exposed to organic solvents were 13 times more likely to give birth to children with major malformations. Specifically noted amongst the detected malformations in the 1999 JAMA study were neuronal migration defects.<sup>20</sup> The article does not specify how many children were born with that exact condition.<sup>21</sup>

For added context, with respect to the neuronal migration diagnosis, it should be noted that an expert neuropsychologist retained by the Anderson family, Dr. Stephen Glass, opined that (1) he does not believe Dalton has "PMG", (2) he believes that Dalton has a "*neuronal migration*" defect, and (3) the different types of brain malformations are simple matter of "*timing*" of the new brain migration as reflected upon the MRI image:

*...I don't believe that it shows PMG. I'm not really sure that it really matters. I have to think about that more, but I -- I think that generically at a minimum we are talking about a **neuronal migration** disorder, and band heterotopias versus polymicrogyria versus any other neuronal migration disorder is as much an issue of timing as an -- as it is an issue of what went on.*

\* \* \*

---

<sup>19</sup> CP 547-76

<sup>20</sup> CP 229-32

<sup>21</sup> *Id.*

*It is probably more in the 20- to 22-week gestation range that polymicrogyria develops, and that's a process of cortical lamination and -- and then sulcaciton, s-u-l-c-a-c-i-t-o-n, and that is the formation of sulci, s-u-l-c-i, and gyri, g-y-r-i, that occurs as a byproduct of that cortical lamination process. That's what's disturbed in polymicrogyria. So it's -- it's all part of a continuous process, but different largely in its timing more than anything.*<sup>22</sup>

And Dr. Stephen Glass noted that the treating physicians are in agreement about the type of malformation from which Dalton suffers:

Q. Can you tell us who Dr. Makari is in relation to Dalton Anderson?

A. Dr. Makari is a child neurologist in Tacoma who along with Dr. Tripp has evaluated Dalton.

Q. Do you have an impression as to what their conclusions were with respect to Dalton Anderson's brain malformations?

A. Both Dr. Tripp and Dr. Makari indicated that this was a neuronal migration disorder.<sup>23</sup>

It should also be noted that Dr. Khattak never agreed that Dalton "likely" suffers from "PMG", as was implied by Akzo Nobel, but only agreed that it was "possible" when deposed by lawyers from Akzo Nobel.

**2. The trial court improperly drew its own conclusions about the meaning of the 1999 JAMA study in relation neuronal migration defects:**

It is true that the 1999 JAMA study did not focus upon neuronal migration defects specifically, but it is not disputed that neuronal

---

<sup>22</sup> CP 577-768 (Exhibit 24 to Declaration of Beauregard (Deposition of Glass Page 9 to 10))

<sup>23</sup> CP 577-768 (Exhibit 24 to Declaration of Beauregard (Deposition of Glass Page 64 to 65))

migration defects were identified within the study.<sup>24</sup> And *Frye* does not and/or should not require proving beyond that which the experts already agree: that organic solvent exposure causes brain malformations/encephalopathy. To the extent which this or any court requires that the more specific type of malformation be verified by an epidemiological study (such as neuronal migration defects), the Anderson family submits that the 1999 JAMA study satisfies this requirement. According to the Anderson family's expert, Dr. Khattak (based upon his extensive experience and review of the medical literature including the 1999 JAMA study which he authored) Dalton's neuronal migration defect was caused by organic solvent exposure.<sup>25</sup>

It is also true that the data underlying the 1999 JAMA study could not be located – now 8 years after publication at Akzo Nobel's request. And in footnote 18 of the response brief, Akzo Nobel once again argues that “*It would be fundamentally unfair to allow him to testify on the basis of the study when the defense has been denied access to the underlying data.*” The trial court accepted Akzo Nobel's contention, and then drew assumptions and presumptions about the meaning of the 1999 JAMA study, including the underlying data, and then determined its own conclusions about how the study related to neuronal migration defects.<sup>26</sup> Based upon the trial court's presumptions about the data underlying the

---

<sup>24</sup> CP 229-32

<sup>25</sup> CP 577-768 (Khattak Deposition)

1999 JAMA study, the opinions of Dr. Khattak and Dr. Glass were excluded.<sup>27</sup>

Put another way, Dalton's case was thrown out despite the fact that the experts for the Anderson family and Akzo Nobel agree that *in utero* organic solvent exposure causes fetal brain malformations. The primary reason for dismissal was because Akzo Nobel wanted to subpoena, peruse, second guess, and argue about meaning of the data underlying the 1999 JAMA study with respect to the specific number of occurrences of neuronal migration defects. The data was not available, so, instead of rummaging through the data underlying the study, the trial court drew its own assumptions about the meaning of the data: "*Because the study stated that 13 of the children born to mothers who had been exposed to organic solvents had 'major malformations' and listed 13 different 'major malformations', the **implication** is that only one of the children born to the mothers in the exposed group showed a neuronal migration defect...*"<sup>28</sup> The trial court's "implication" approach to *Frye* cannot possibly be the law — the act of second guessing the experts' data underlying epidemiological studies. This Court has already explained that "judges do not have expertise to decide whether a challenged scientific theory is

---

<sup>26</sup> CP 779-91

<sup>27</sup> *Id.*

<sup>28</sup> CP 779-791



correct...” *State v. Cauthron*, 120 Wash. 2d 979, 887, 846 P.2d 502

(1993). Under analogous circumstances, another court explained:

For the district court to seize on the putative flaws of studies favorable to plaintiff, and then to privilege certain studies favorable to the defendant, was impermissibly to place a thumb on defendant’s side of the scale and to encroach on the jury’s prerogative to weigh the relative merits and credibilities of competing studies ... Thus, to the extent that none of the studies is flawless or dispositive, their relative merits seems to us to be a classic question for the jury. Trial courts should not arrogate the jury’s role in “evaluating the evidence and the credibility of expert witnesses” by “simply cho[o]s[ing] sides in [the] battle of the experts.”

*Christophersen v. Allied-Signal Corp.*, 902 F.2d 362, 366 (5<sup>th</sup> Cir. 1990).

In this case, the trial court did exactly that: decided the scientific meaning of the 1999 JAMA study and choose Akzo Nobel’s expert’s testimony over that of the Anderson family’s.

In *Berry*, the trial court followed a similarly flawed line of reasoning with respect to epidemiological studies, and the Florida Supreme Court explained “the trial court’s...reasoning for denying admissibility-that the underlying epidemiological studies were equivocal as to causation-we find that the trial court ultimately misunderstood the nature of epidemiological studies and was unnecessarily concerned that the studies did not prove causation.” 709 So. 2d at 567. The *Berry* Court elaborated: “epidemiological studies do not fix the cause-they merely demonstrate the probabilities of cause...However, epidemiological studies themselves are not designed to demonstrate whether a particular agent did cause the disease, and the trial court erred in concluding that the studies

were unreliable because they failed to establish a causal relationship.” *Id.* at 568.<sup>29</sup> In this case, the trial court erred in the same way.

Other courts have analyzed and rejected the notion that a claimant must come forward with a pin-point perfect epidemiological study supportive of the precise causation theory at issue. *See e.g. Heller v. Shaw Industries, Inc.*, 167 F.3d 146 (3<sup>rd</sup> Cir. 1999); *Kennedy v. Collagen Corp.*, 161 F.3d 1226, 1229 (9<sup>th</sup> Cir. 1998) (finding district court abused its discretion by excluding expert testimony that was based upon reliable methodology simply because “no epidemiological or animal studies” linked defendant’s product to plaintiff’s disease). In *Heller*, the Court explained that “we do not believe that a medical expert must always cite published studies on general causation in order to reliably conclude that a particular object caused a particular illness.” *Id.* at 155. The *Heller* Court further explained that

To do so would doom from the outset all cases in which the state of research on the specific ailment or on the alleged causal agent was in its early stages, and would effectively resurrect a *Frye*-like bright line standard, not by requiring that a methodology be “generally accepted,” but by excluding expert testimony not backed by published (and presumably peer-reviewed) studies. We have held that the reliability analysis applies to all aspects of an expert’s testimony: the methodology, the facts underlying the expert’s opinion, the link between the facts and the conclusions...However, not only must each stage of the expert’s testimony be reliable, but each stage must be evaluated practically and flexibly without bright-line exclusionary (or inclusionary) rules.

---

<sup>29</sup> On page 14 of the responsive brief, Akzo Nobel misleadingly contends that “*The article did not establish a causal link between the chemicals and conditions at issue in this case, not matter how broadly defined by Appellants.*”

*Id.*; see also *McCulloch v. H.B. Fuller Co.*, 61 F.3d 1038 (2<sup>nd</sup> Cir. 1995) (affirming admission of treating doctor's testimony despite the fact that he "could not point to a single piece of medical literature that says glue fumes cause throat polyps.") In another toxic tort case, the Supreme Court of Idaho explained:

As long as the basic methodology employed to reach such a conclusion is sound...[the] law does not preclude recovery until a "statistically significant" number of people have been injured or until science has had the time and resources to complete sophisticated laboratory studies...

*Earl v. Cryovac, A Div. of W.R. Grace Co.*, 115 Idaho 1087, 1095, 772 P.2d 725 (1989), quoting, *Ferebee v. Chevron Chemical Co.*, 736 F.2d 1529, 1536 (D.C. Cir. 1984); see also *Callahan v. Cardinal Glennon Hosp.*, 863 S.W. 2d 852 (Mo. 1983) (plaintiff experts were allowed to testify that failure to properly treat an abscess three weeks after infant received polio vaccine resulted in suppression of immune system and infant's contraction of paralytic polio; court held causation evidence was sufficient even though experts did not base their opinion on epidemiological studies).

Moreover, even accepting, without agreeing, for a moment that the trial courts should be scrutinizing the data underlying epidemiological studies (and the testimony experts and authors that researched and wrote them) the trial court's conclusion by "implication" about the probabilities demonstrated by of the 1999 JAMA study with respect to neuronal migration disorders is highly questionable. In the response briefing, Akzo Nobel points out that brain malformations of this nature typically occur

*“in at least 1 out of every 2,500” births.*<sup>30</sup> In the JAMA study *at least 1* of the 125 mothers in the control group gave birth to a child with a neuronal migration defect. Based upon this knowledge about the data and statistical occurrence of neuronal migration defects, the trial court explained that *“this court would need additional information to determine whether that one event was significant.”*<sup>31</sup>

The Anderson family submits that the probability that even 1 of the 125 babies born to a mother exposed to organic solvents in the workplace from the 1999 JAMA study was born with a neuronal migration defect is statistically significant when weighed against the fact that, generally speaking, the condition is pronounced in only 1 out of 2500 births. It is also important to note that even if there were only 1 instance of neuronal migration defects detected in the 1999 JAMA study, this is one major malformation amongst a constellation of 13 other major malformations.<sup>32</sup> By comparison, the study’s control group demonstrated only 1 major malformation, and 0 neuronal migration defects.<sup>33</sup> In lieu of analyzing and/or recognizing this statistical improbability, the trial court threw out Dalton’s case against Akzo Nobel. The trial court in this case committed a similar error to that of the trial court in *Berry*.

---

<sup>30</sup> Akzo Nobel’s Response Brief, Page 28.

<sup>31</sup> *Id.*

<sup>32</sup> CP 229-32

<sup>33</sup> *Id.*

Additionally, according to Washington law, the *Frye* test only prohibits expert testimony “[i]f there is significant dispute between the qualified experts as to the validity of the scientific evidence...” *Cauthron*, 120 Wn. 2d at 887. The Anderson family submits to this Court that the differing opinions about the strength with which the 1999 JAMA study demonstrated an association between fetal organic solvent exposure and neuronal migration defects specifically (versus major malformations generally) certainly does not constitute a “significant dispute” between the experts. This is particularly true in that the experts agree that organic solvent exposure causes brain malformations generally, and they also agree upon the methodology for making a causative assessment. And because this dispute between the experts is not “significant”, the dismissal based upon the strength of the association which is demonstrated by the 1999 JAMA study was error.

**3. The Anderson family also relies upon a 2004 study authored by Dr. Koren which is determinative that *in utero* organic solvent exposure causes cognitive delays:**

In addition to the 1999 JAMA study, the Anderson family also submitted another publication authored by Dr. Koren from 2004: *Child Neurodevelopmental Outcome and Maternal Exposure to Solvents*. According to Akzo Nobel, this article from 2004 “*identified an association with [in utero organic solvent exposure] and mild cognitive or language problems.*”<sup>34</sup> Akzo Nobel claims that this 2004 article is

---

<sup>34</sup> Akzo Nobel Response Brief, Page 30

distinguishable because cognitive problems “*were not associated with Dalton Anderson.*”<sup>35</sup> Akzo Nobel’s assertion is flat out untrue. Dalton suffers from cognitive delays.<sup>36</sup> Therefore, the article from 2004 which was authored by Dr. Koren is conclusive under *Frye*: *in utero* organic solvent exposure causes cognitive delays.

In an attempt to distinguish this 2004 study, at the trial court level, Akzo Nobel argued that the *specific degree of cognitive delay* suffered by Dalton was more severe than that suffered by the children in the study. In other words, according to Akzo Nobel, the 2004 study is not determinative under *Frye* because Dalton’s delays are *really* bad versus just *sort of* bad. First of all, this is a distinction without a difference. Second of all, there is no objective data indicating that Dalton’s cognitive delays are any different than those suffered by the children in the 2004 study.<sup>37</sup> The 2004 study establishes under *Frye* that it is generally accepted in the scientific community that *in utero* exposure to organic solvents causes cognitive delays such as those suffered by Dalton. This study cannot simply be ignored and has not been distinguished by Akzo Nobel in any meaningful way.

---

<sup>35</sup> *Id.*

<sup>36</sup> CP 455-91

<sup>37</sup> CP 455-91

**4. Applying *Frye* in the manner which is urged by Akzo Nobel places and unrealistic and even unethical burden upon the Anderson family:**

Adherence to *Frye* in the manner which requires proving that the *specific* causation theory itself is generally accepted in the scientific community places an insurmountable and unrealistic burden upon litigants such as the Anderson family. For example, in this case, *if* the Anderson family came forward with a scientific study proving with absolute meta-physical-certitude that neuronal migration defects were caused by organic solvent exposure, then Akzo Nobel's next *Frye* hearing noted by Akzo Nobel's clever lawyers would be to argue that the Anderson family did not come forward and prove that the sub-group of neuronal migration defects suffered by Dalton has been linked to organic solvent exposure. The next *Frye* motion after that would be about the cell structures of neuronal migration defects. And the next *Frye* motion after that would be about mitochondria of the cell structures of neuronal migration defects. And so on, and so on, and so on. Requiring litigants, and their experts, to come forward with pinpoint perfect studies upon human subjects (babies in this instance) proving with statistical perfection the causation correlation identical to that which is suffered by the claimant is not plausible.

Another problem with applying *Frye* in the manner proposed by Akzo Nobel is illustrated by the attempt to distinguish the 2004 study which was authored by Dr. Koren. In relation to that study, Akzo Nobel concedes that it establishes that *in utero* organic solvent exposure causes cognitive delays in children. However, in an effort to distinguish that

study, Akzo Nobel argues about the purported difference between Dalton's cognitive delays and those of the children in the study. Evidently, Akzo Nobel would require the Anderson family to come forward with a study proving that other children had the exact same IQ as Dalton, and not just that the children suffered with cognitive delays generally as a result of organic solvent exposure. Again, this level of exacting detail is not and cannot possibly be what this Court expects of litigants when applying *Frye* to the facts of any given case.

Beyond that, in this instance, we are also talking about the malformation of a human baby's brain. In order to meet the *Frye* standard which is urged by Akzo Nobel and was applied by the trial court, the Anderson family must find an expert who exposes pregnant mothers to organic solvents, and who is willing to expose enough pregnant mothers to organic solvents in order to research and write a paper about neuronal migration defects. The *Berry* Court described the process of doing so as follows:

To establish that a given substance was a necessary causal link to the development of an individual's disease, in theory a scientist might obtain reliable information by engaging in experimental studies with human beings. For example, to determine whether exposure to a certain level of suspected toxin is associated with a particular disease, the scientist might compare two randomly selected groups of people. One of the groups would be exposed to certain doses of the toxin over a prescribed length of time and the other group would not.

*Berry*, 709 So. 2d at 557. Surely, the law and the *Frye* test was not intended to promote conducting tortuous experiments of this nature upon



human subjects, and embryos. In that regard, a distinguished neurologist, Dr. Glass, opined that such “causation” research as applied to pregnant mother and babies would be unethical: *“that is to say who can possibly do a prospective study of Toluene or Xylene or other organic solvent exposures responsibly? We can’t. We wouldn’t – we wouldn’t subject a pregnant mother to have that happen.”*<sup>38</sup> “Because of these ethical prescriptions, rather than experimental methods, epidemiologists use observational methods to study persons exposed to a suspected toxic substance to determine whether an association exists between exposure to the chemical and the development of a disease.” *Berry*, 709 So. 2d at 557.

It should also be noted that the 1999 JAMA study is the closest that science is every likely to come to evaluating whether or not organic solvents cause specific types of brain malformations.<sup>39</sup> The “data” from the 1999 JAMA study consisted of live pregnant mothers that worked around organic solvents who voluntarily provided details of their exposures and pregnancies to Dr. Khattak and Dr. Koren.<sup>40</sup> A prospective study of live individuals is rare, and a study focusing upon the existence of a particular brain malformation, be it a neuronal migration defect or PMG, would be virtually unheard of and of limited utility to the scientific community. Moreover, the 1999 JAMA study *did* detect the existence of neuronal migrations defects amongst the group of pregnant mothers

---

<sup>38</sup> CP 577-768 (Exhibit 24 to Declaration of Beauregard (Glass Deposition Pages 15))

<sup>39</sup> CP 229-32

exposed to organic solvents within the workplace.<sup>41</sup> But according to Akzo Nobel, this prospective study involving human pregnant mothers and children is simply not good enough to meet the *Frye* test. The Anderson family submits that a more exacting requirement is not realistic. And the law and *Frye* test cannot possibly be this unforgiving.

**5. Adhering to the “methodology” based application of the *Frye* test allows Courts to avoid deciding between the conflicting conclusions of the experts:**

This Court can avoid the slippery slope of determining the proper diagnostic detail required for the *Frye* test altogether by following the trend from other jurisdictions away from scrutinizing the specific scientific conclusion of the experts, and focusing primarily, or exclusively, on the general acceptance of the underlying methodology employed.<sup>42</sup> This Court already suggested this inclination in *Gregory*: “[o]nce a methodology is accepted in the scientific community, the application of the science to a particular case is a matter of weight and admissibility under ER 702, which allows qualified expert witnesses to testify if scientific, technical, or other specialized knowledge will assist the trier of

---

<sup>40</sup> *Id.*

<sup>41</sup> *Id.*

<sup>42</sup> See *In re Commitment of Simons*, 213 Ill.2d 523, 290 Ill.Dec. 610, 821 N.E.2d 1184 (2004)citing *Donaldson v. Central Illinois Public Service Co.*, 199 Ill.2d 63, 77-79, 262 Ill. Dec. 854, 767 N.E.2d 314 (2002) (“The *Frye* test applies only to “new” or “novel” scientific methodologies” and “generally speaking, a scientific methodology is considered “new” or “novel” if it is “original or striking” or ‘does not resembl[e] something formerly known or used.’”); *State v. Baby*, 404 Md. 220, 946 A.2d 463 (2008)(Stating that *Frye* hearing is needed if a “new scientific technique’s validity is in controversy in the relevant scientific community.”); *Grady v. Frito-Lay, Inc.*, 576 Pa. 546, 558-61, 839 A.2d 1038 (2003)(Proponent is not required to “prove that the scientific

fact.” 158 Wn. 2d at 829-30. And it must be noted that, quite ironically, the trial court which rendered the *Frye* ruling in this case has subsequently offered rulings embracing the methodology based approach and lending essentially *no* scrutiny to the causation opinion of the conflicting experts themselves. A copy of such an opinion dated February 12, 2009 from the underlying trial court is attached as an Appendix hereto as *Peterson v. Dillion*, King County Cause No. 06-2-25543-7.

By contrast, in this case, the underlying trial court relied heavily upon a case from Division III, *Grant v. Boccia*, 133 Wn. App. 176, 137 P.3d 20 (2006), and placed great emphasis upon requiring proof that “the causation opinion itself” be generally accepted by the scientific community even if the underlying methodology was readily established. *Grant* was a case involving the causation of fibromyalgia by way of automobile accidents and, based upon *Frye*, Division III rejected the causation premise: “*Until medical science determines with sufficient reliability and acceptance that a casual relationship exists between trauma and fibromyalgia, such evidence is inadmissible under the Frye test as adhered to in this state.*” *Id.* at 185-6.

In comparison, in *Peterson v. Dillion*, the same underlying trial court which ruled upon this case indicated that “the *Grant* court did not even address what the relevant scientific community has generally accepted to be reliable methodologies for determining medical

---

community has also generally accepted the expert’s conclusion” but that proponent must

causation.”<sup>43</sup> And then, based upon a methodology focused application of the *Frye* test, the same trial court rendered a ruling which, if affirmed, would overrule *Grant* upon review in Division I. Put another way, premised upon the methodology based application of the *Frye* test, the trial court ruled that the theory of fibromyalgia being caused by automobile accidents was generally accepted in the scientific community, or at least the underlying methodology was sound.

**6. Akzo Nobel does not dispute that the applicable methodology has been generally accepted by the scientific community, and that methodology was appropriately applied with respect to determining the cause of Dalton’s brain malformations:**

To the extent that this Court elects to continue to apply *Frye*, it should continue embrace the methodology focused application of the test, and it should so do in this case. In 1993, this Court explained that

It is important to distinguish, however, between the general acceptance of the methodology, and the acceptance of the results of a particular study...If the particular technique is sufficiently accepted in the scientific community at large, any remaining concerns about the possibility of error or mistakes being made in the case at hand can be argued to the fact finder.

*State v. Cauthron*, 120 Wash 2d, 879, 889, 846 P.2d 502 (1993).

According to another case from Florida Supreme Court:

[U]nder *Frye*, the inquiry must focus only on the general acceptance of the scientific principles and methodologies upon which an expert relies in rendering his or her opinion.

---

show that the methodology has been generally accepted).

<sup>43</sup> Page 13 of Trial Court Order Attached as Appendix.

\* \* \*

Trial courts must resist the temptation to usurp the jury's role in evaluating the credibility of experts and choosing between legitimate but conflicting scientific views.

*Marsh*, 977 So. 2d at 549 (allowing expert testimony about the cause of fibromyalgia premised upon accepted methodology).

In this case, Akzo Nobel does not dispute that the determinative methodology is generally accepted in the medical community. Akzo Nobel never offered any contradictory evidence or argument in that regard. Instead, Akzo Nobel strains to take issue with the manner in which the particulars, such as which medical records Dr. Khattak reviewed or how long he interviewed Ms. Anderson. These are not issues relevant to the *Frye* analysis, did not persuade the trial court, and are, in large part, inaccurate representations of the record on the part of Akzo Nobel.

In that regard, Akzo Nobel makes erroneous claims, such as that found in footnote 6 of the response brief, stating that “*There has been no evidence that Ms. Anderson worked with solvents for anything even close to this duration.*” In truth, Ms. Anderson and a coworker, Laurinda Rowland, have vivid recollections of Ms. Anderson spending voluminous period of time handling organic solvents and supportive evidence is within the record.<sup>44</sup> Akzo Nobel also makes the erroneous claim that Ms. Anderson did not report symptomology to her treating doctors. In truth, the available medical records reflect that Ms. Anderson did report

---

<sup>44</sup> CP 68-9; 437-8; 103-52;455-91

exposure related symptomology to her treating physicians.<sup>45</sup> And Akzo Nobel makes the erroneous claim that Dr. Khattak did not properly rule out “genetics” as a possible cause of Dalton’s brain malformations. In truth, Dr. Khattak relied up on the “genetics” testing which was conducted by the treating physicians and ruled out any such abnormality.<sup>46</sup> In other words, the contrary representations on the part of Akzo Nobel are simply untrue.

#### **7. Conclusion:**

The trial court committed error in this matter by tossing out Dalton’s case against Akzo Nobel premised upon purported flaws in the data underlying the epidemiological studies supporting the Anderson family’s claims. Dalton suffers from brain damage and the corresponding cognitive delays as a result of *in utero* organic solvent exposure. The studies relied upon by the Anderson family support the premise that organic solvent exposure causes major fetal malformations including of the brain and specifically neuronal migration defects. An epidemiological study that was authored by the defense’s expert, Dr. Koren, linked cognitive delays, such as that which is suffered by Dalton, and organic solvent exposure during pregnancy. In reaching the conclusions expressed in this case, both Dr. Khattak and Dr. Glass relied upon the accepted and established methodology for determining whether or not *in utero* organic

---

<sup>45</sup> CP 455-91

solvent exposure caused Dalton's condition. Premised upon the evidence of record, the trial court should be reversed with respect to the application of the *Frye* test.

**C. The provisions set forth under RCW 49.17.160 do not provide an "adequate" legal mechanism upon which to obtain relief for a retaliatory discharge.**

Akzo Nobel also now argues that RCW 49.17.160 provides an "adequate" remedy for obtaining redress from a retaliatory discharge. As is set forth herein, the remedies afforded under RCW 49.17.160 are not adequate. Even if a complainant employs the procedure set forth under RCW 49.17.160, that same complainant ends up in the same place with or without WISHA by his or her side: litigating the retaliatory discharge claim in the superior court. And so it follows that Akzo Nobel's arguments are not correct, and the trial court erred in that regard.

**1. The arguments being advanced by Akzo Nobel are without merit in that the determinative issue is whether or not the complainant has an opportunity to obtain complete redress before a tribunal that is empowered to grant complete relief.**

Akzo Nobel now relies predominantly upon *Korslund v. Dyncorp Tri-Cities Services, Inc.*, 156 Wn.2d 168, 125 P.3d 119 (2005) as being purportedly determinative as to the availability of a private cause of action for violation of public policy in the context which is presently before the Court. In so doing, Akzo Nobel argues that, by analogy, based upon the

---

<sup>46</sup> Subsequent testing conducted by Akzo Nobel's experts also failed to establish any "genetic" abnormality which could be associated with Dalton's condition. See CP 818-

principles and law set forth in *Korslund*, that there “other means of promoting the public policy [which] are adequate so that recognition of the tort claim...is unnecessary to protect the public policy.” *Id.* at 183. At face value, Akzo Nobel’s arguments seem plausible. Upon proper scrutiny, the issues as between *Korslund* and this case are very distinct, and it is apparent that Akzo Nobel’s arguments have no merit in this context.<sup>47</sup>

In *Korslund*, the Supreme Court noted that under the Energy Reorganization Act (ERA) which address issues of national concern in relation to nuclear energy “provides an **administrative process** for adjudicating whistleblower complaints, and provides for order to the violator to take ‘affirmative action to abate the violation;’ reinstatement of the complainant to his or her former position with the same compensation, terms, conditions of employment; back pay; compensatory damages; and attorney and expert witness fees.” *Id.* at 182, citing, 42 U.S.C. 5851(b)(2)(B) (emphasis added). The “administrative process” is adjudicative before an Administrative Law Judge and allows for the complainant to have immediate access to a forum wherein to remedy the dispute. *Id.* In other words, under the ERA, the complainant is guaranteed a day before an objective tribunal which is empowered to

<sup>47</sup> The arguments that are offered by Akzo Nobel are tantamount to saying: “*these statutes look kind of alike so the results must be the same.*” The fundamental flaw in Akzo Nobel’s argument is that there is no substantive or qualitative analysis provided as to why the law is what it is or why, as a matter of principle and legal mechanics, the Court reached the result that was reached in *Korslund*.



bestow complete relief upon the proper showing of proof that discrimination against public policy has occurred. *Id.*

By contrast, the whistleblower protections provided in this context under RCW 49.17.160 (WISHA) are very different in that adjudicative relief *is not* provided by an “administrative process” as compared to those which are provided under the ERA. Under RCW 49.17.160, WISHA *is not* empowered to render adjudicative determinations and order comprehensive relief to a complainant. Instead, RCW 49.17.160 simply requires that WISHA conduct an investigation, and if in WISHA’s opinion a violation has occurred, then WISHA is required to (“shall”) “bring an action in the superior court of the county wherein the violation occurred.” *Id.*

In essence, RCW 49.17.160 solely requires that WISHA assist the complainant in superior court if, after an investigation it seems likely the law has been broken. Additionally, even if WISHA disagrees with the complainant, the very language of the statute still permits redress in superior court but without affording the complainant with the benefit of WISHA’s litigation resources and assistance. *Id.* The most important distinction as between the ERA and RCW 49.17.160 is that under the ERA an administrative law judge conducts and adjudication on the merits and can provide the complainant complete relief, whereas under RCW 49.17.160, at best, the complainant only gets the benefit of added litigation resources in superior court. In other words, in essence, Ms. Anderson

could have acquired a "litigation buddy" and nothing more. Based upon this critical distinction, *Korshund* is readily distinguishable, and Akzo Nobel's argument in relation to purportedly "adequate" remedies for perpetuating the public policy favoring protecting aggrieved whistleblowers in the context which is not before the Court fails.

**2. Under the federal law, there is no forum before which Ms. Anderson could appear in order to obtain relief from and against Akzo Nobel.**

The federal counterpart under the Occupational Safety and Health Act (OSHA) to that of WISHA (RCW 49.17.160) similarly only allows for a preliminary investigation conducted on the part of the OSHA authorities, and then subsequent litigation and potential redress in district court. *See* 29 U.S.C. 660.<sup>48</sup> In other words, unlike the formal adjudicative process as was available to the Hanford Nuclear Plant employees in *Korshund*, there is/was no administrative adjudicative body for Ms. Anderson to rely upon and appear before in order to compel Akzo Nobel to compensate her for the wrongful termination. At best, under the controlling laws, Ms. Anderson could have received the benefit of having OSHA and/or WISHA in her corner of the ring during litigation against Akzo Nobel. But no matter what, Ms. Anderson is forced to obtain redress in Court with, or without, the assistance of OSHA and/or WISHA.

---

<sup>48</sup> Employees have no private right of action under OSHA, since Congress did not intend that Secretary's prosecutorial discretion should be subordinated to rights of employees; only right employees have in enforcement proceedings is limited right to challenge reasonableness of time fixed in citation for abatement of violation. *Donovan v. OSHRC*, 713 F2d 918 (2<sup>nd</sup> Cir. 1983). And so it follows that the only jurisdiction whereabouts Ms. Anderson can obtain redress while maintaining control over the proceedings is in superior court.

**3. The proper focus is upon whether or not WISHA is empowered to grant relief for discrimination that violates public policy; WISHA has no such power.**

The operative question is focused upon what “relief” is available under the law, but then misapplies those principles to the facts of this case. Under RCW 49.17.160, WISHA is not empowered to grant any relief to Ms. Anderson. Instead, according to the letter of the statute:

...the **superior court** shall have jurisdiction, for cause shown, to restrain violations of subsection (1) of this section and order all appropriate relief including rehiring or reinstatement of the employee to his former position with back pay.

*Id* (emphasis added).<sup>49</sup> Put another way, under RCW 49.17.160, no power to grant relief is vested in WISHA other than as an investigator and advocate. Whereas the superior courts are the proper tribunal before which Ms. Anderson’s dispute must be litigated. As was previously pointed out, under OSHA, the result is no different, and redress must be obtained in open court rather than before an administrative tribunal.

In light of the clear language under RCW 49.17.160, and the OSHA counterpart too, Akzo Nobel’s argument concerning purportedly adequate “relief” under those laws is not correct. The very statutes being relied upon expressly require that complainants, such as Ms. Anderson, to obtain compulsory redress against their employers in Court. In this instance, though Akzo Nobel would like this claim to simply vanish with

---

<sup>49</sup> In comparison, in *Korslund*, under the ERA, it could be ordered that “the person who committed such violation to...reinstate the complainant to his former position together with the compensation (including back pay), terms, conditions, and privileges of this employment...”

no further adjudication on the merits, Ms. Anderson has no other alternative but to file a claim in superior court. There is no other place else for Ms. Anderson to take her claim to obtain relief.

**4. By Legislative enactment and as is confirmed by case law, Ms. Anderson has a right to obtain redress in superior court.**

As is expressly stated within RCW 49.17.160, the Legislature enacted the right to bring a private cause of action for retaliatory discrimination correlating with safety in the workplace. And it should be noted that Division I of the Court of Appeals has previously determined that RCW 49.17.160 *is not* the “exclusive” or “mandatory” remedy to whistleblowers in Washington, and that private causes of action are permitted under the law. *Wilson v. The City of Monroe*, 88 Wn. App. 113, 943 P.2d 1134 (1997) (holding that private cause of action available under RCW 49.17.160). In light of the Legislative dictates set forth in RCW 49.17.160 coupled with Court of Appeals holding in *Wilson*, Ms. Anderson is permitted to pursue a private right of action premised upon the unlawful retaliatory discharge which occurred based upon her having complained to WISHA.

///

///

///

///

## V. CONCLUSION

For the reasons set forth herein, the trial court should be reversed on the issues of comparative fault, the application of the Frye test, and the retaliatory discharge requirements set forth under RCW 49.17.160.

RESPECTFULLY SUBMITTED this 13 day of March, 2009.

CONNELLY LAW OFFICES

By

John R. Connelly, Jr., WSBA #12183  
Lincoln C. Beauregard, WSBA #32878  
Attorneys for the Anderson family

RECEIVED  
SUPREME COURT  
STATE OF WASHINGTON

2009 MAR 13 P 2:56

BY RONALD R. CARPENTER

---

King County Superior Court No. ~~07-2-10209-4~~

---

WASHINGTON STATE SUPREME COURT

---

Julie Anderson, individually and on behalf of Dalton Anderson, a minor, and  
Darwin Anderson,

Appellant,

vs.

Akzo Nobel Coatings, Inc. and Keith Crockett,

Respondents

---

**APPENDIX RE: *Peterson v. Dillon*, King County Superior Court Cause No.  
06-2-25543-7 Order dated February 12, 2009**

---

John R. Connelly, Jr, WSBA No. 12183  
Lincoln C. Beauregard, WSBA No. 32878  
CONNELLY LAW OFFICES  
2301 N. 30<sup>th</sup> Street  
Tacoma, Washington 98403  
(253) 593-5100  
Fax (253) 593-0380

 ORIGINAL

FILED  
09 FEB 12 PM 4:36  
KING COUNTY  
SUPERIOR COURT CLERK  
KENT, WA

ORIGINAL

SUPERIOR COURT OF WASHINGTON FOR KING COUNTY

TINA C. PETERSON,

Plaintiff,

vs.

JOHN G. DILLON et al.,

Defendants.

No. 06-2-25543-7 SEA

ORDER DENYING DEFENDANTS'  
MOTION IN LIMINE TO EXCLUDE  
CAUSATION TESTIMONY RE.  
HYPERMOBILITY JOINT SYNDROME  
AND FIBROMYALGIA

THIS MATTER came on for hearing before the undersigned on defendant's motion in limine to exclude the testimony of plaintiff's experts regarding the role of trauma in causing or triggering the plaintiff's fibromyalgia or hypermobility joint syndrome. The court considered all pleadings filed by the parties in connection with the motion, including the declarations and the extensive attachments thereto, including the many medical articles, depositions and preservation depositions, oral testimony from a number of different expert witnesses called by the parties, and the exhibits submitted in the 2-day evidentiary hearing in this matter. Being fully advised, it is now hereby

ORDERED that the defendant's motion is DENIED.

1 DISCUSSION

2 Defendant relies on *Grant v. Boccia*, 133 Wn. App. 176, 178 (2006), *review denied*,  
3 159 Wn.2d 1014 (2007), in which Division Three of the Court of Appeals held that a plaintiff  
4 could not present expert testimony at trial that his fibromyalgia had been proximately caused  
5 by a motor vehicle collision with the defendant, because the court found that the theory that  
6 fibromyalgia can be caused by trauma was controversial and not generally accepted in the  
7 relevant scientific community. The *Grant* court concluded that expert testimony regarding the  
8 cause of the plaintiff's fibromyalgia did not meet the *Frye*<sup>1</sup> standard in Washington.

10 The court in *Grant* began its analysis by citing the well-established criteria that courts  
11 must consider before admitting novel scientific evidence: "(1) whether the scientific principle  
12 or theory from which the testimony is derived has garnered general acceptance in the relevant  
13 scientific community under the *Frye* standard; and (2) whether the expert testimony is properly  
14 admissible under ER 702." *Grant* at 178, citing *State v. Riker*, 123 Wn.2d 351 (1994).

16 In examining a *Frye* question, the court must determine: "(1) whether  
17 the underlying theory is generally accepted in the scientific community  
18 and (2) whether there are techniques, experiments, or studies utilizing  
19 that theory which are capable of producing reliable results and are  
20 generally accepted in the scientific community." Under the *Frye* test,

21 <sup>1</sup> *Frye v. United States*, 293 F. 1013 (D.C. Cir. 1923). In *Frye*, a criminal defendant tried to introduce evidence  
22 from a polygraph or "lie detector" test to corroborate his exculpatory statements. The District of Columbia Court  
23 of Appeals excluded the evidence, because the test had not gained "general acceptance" among authorities in the  
24 fields of physiology and psychology. The court stated:

25 Just when a scientific principle or discovery crosses the line between the experimental and  
26 demonstrable stages is difficult to define. Somewhere in this twilight zone the evidential force  
of the principle must be recognized, and while courts will go a long way in admitting expert  
testimony deduced from a well-recognized scientific principle or discovery, the thing from  
which the deduction is made must be sufficiently established to have gained general accep-  
tance in the particular field in which it belongs.

293 F. at 1014.



1 we do not determine if the scientific theory underlying the proposed  
2 testimony is correct. Rather, we must look to see whether the theory has  
3 achieved general acceptance in the appropriate scientific community.

4 *Grant* at 179 (citations omitted).

5 Washington has adopted the *Frye* test for evaluating the admissibility of  
6 new scientific evidence. *State v. Cauthron*, 120 Wn.2d 879, 886 (1993)  
7 (citing *Frye v. United States*, 293 F. 1013, 1014 (D.C. Cir. 1923)). The  
8 primary goal is to determine “whether the evidence offered is based on  
9 established scientific methodology.” *State v. Gore*, 143 Wn.2d 288, 302  
10 (2001). Both the scientific theory underlying the evidence and the  
11 technique or methodology used to implement it must be generally  
12 accepted in the scientific community for evidence to be admissible under  
13 *Frye*. *Id.* “If there is a *significant* dispute among *qualified* scientists in  
14 the relevant scientific community, then the evidence may not be admit-  
15 ted,” but scientific opinion need not be unanimous. *Id.*

16 *State v. Gregory*, 158 Wn.2d 759, 829-830 (2006) (citations and emphasis in original). “How-  
17 ever, the *Frye* test is unnecessary if the evidence does not involve new methods of proof or  
18 new scientific principles.” *In re Detention of Halgren*, 156 Wn.2d 795, 806 (2006). “[T]he  
19 core concern . . . is only whether the evidence being offered is based on established scientific  
20 methodology.” *In re Detention of Post*, 145 Wn. App. 728, 755-757 (2008), quoting from *In*  
21 *re Detention of Thorell*, 149 Wn.2d 724, 754 (2003), *cert. denied*, 541 U.S. 990 (2004).

22 Although courts in Washington and elsewhere have addressed challenges to admissi-  
23 bility of evidence under *Frye* largely on an *ad hoc* basis, analysis of the case law indicates that  
24 in determining the applicability of the *Frye* standard, a court must consider the following three  
25 issues:

- 26 1. What is the “relevant scientific community”?
2. What is the “scientific theory” whose general acceptance in the relevant scientific community must be shown?

1                   3. Is the proffered scientific evidence based upon "established scientific  
2                   methodology"?

3                   A. WHAT IS THE "RELEVANT SCIENTIFIC COMMUNITY"?

4                   This court was unable to find any discussion or analysis – either in Washington juris-  
5                   prudence or in the opinions from other state courts that utilize a *Frye* standard for admission of  
6                   scientific evidence – as to how a court should define or determine which particular scientific  
7                   community is the "relevant scientific community" whose general acceptance is at issue during  
8                   a *Frye* hearing. This case squarely presents that question.

9                   In addition to submitting numerous scientific articles, the parties presented testimony  
10                  from numerous expert witnesses. Each of the plaintiff's medical experts specializes in treating  
11                  patients who suffer from fibromyalgia, hypermobility joint syndrome, and/or chronic wide-  
12                  spread pain, and most of them are actively engaged in doing research on these conditions. All  
13                  of plaintiff's medical experts testified that, among the physicians who are so engaged, it is  
14                  generally accepted that trauma can cause or trigger fibromyalgia in certain people whose pre-  
15                  existing condition makes them susceptible to developing it.

16                  Andrew Holman, M.D., a board certified rheumatologist who does research in fibro-  
17                  myalgia and chronic pain, treats patients with these disorders, and who met Ms. Peterson on  
18                  two occasions and interviewed her at length, testified at the *Frye* hearing. It was his opinion  
19                  that Ms. Peterson's symptoms of chronic widespread pain were due at least in part to referred  
20                  pain from compression of her cervical spine, which resulted from injury to her joints and  
21                  ligaments caused by the motor vehicle collision. Dr. Holman also opined that there is strong  
22                  consistency in current medical research indicating that fibromyalgia is an "autonomic dys-  
23                  regulatory state," meaning that patients with fibromyalgia process pain perception differently  
24                  25                  26

1 from healthy people, and that their brains amplify otherwise minor sensations into sensations  
2 of pain.

3 Dr. Holman noted that in his own research on patients with chronic widespread pain,  
4 87% of them had compression of their spinal cord when their necks were extended. According  
5 to Dr. Holman, who regularly attends rheumatology conferences where the subject is dis-  
6 cussed, there is a general consensus among the community of rheumatologists who are trained  
7 and experienced in caring for patients with fibromyalgia or chronic widespread pain, that  
8 trauma is a cause of chronic widespread pain in some people. He noted a growing consensus  
9 that fibromyalgia should be viewed not as a 'disease', but as a mechanism for chronic pain,  
10 caused by lack of Stage IV sleep and chronic compression of the cervical spine. In support of  
11 this theory, Dr. Holman opined that chronic widespread pain and symptoms of fibromyalgia  
12 have been elicited in experimental animals from discrete intermittent compression of the spinal  
13 cord.  
14  
15

16 Plaintiff also presented the testimony of Rodney Graham, M.D., who has practiced  
17 rheumatology since 1969, and who is internationally recognized in his field. Dr. Graham has  
18 served as the president of the British Society of Rheumatology, is a member of the American  
19 College of Rheumatology, and has been an editor of peer-reviewed journals in rheumatology.  
20 He has the British equivalent of board certification in rheumatology, and is internationally res-  
21 pected, having earned honors in his field in Great Britain, France, Russia, and the Czech  
22 Republic. His specialty in rheumatology since the 1960's has been on inheritable disorders of  
23 connective tissues, including hypermobility joint syndrome.  
24  
25  
26

1 Joint hypermobility is an inherited condition in which a person's joints have a wider  
2 range of motion than does the normal population. The condition is not in and of itself painful,  
3 and many dancers and athletes have this condition. Ms. Peterson has it. According to Dr.  
4 Graham, hypermobility as a pre-existing condition makes people more susceptible to injury,  
5 because their ligaments are more elastic and do not provide as much protection to their joints  
6 as do those of non-hypermobile persons. The greater the laxity of ligaments, the less stability  
7 they provide to joints. Ligaments are made of collagen, and people with hypermobile joints  
8 have a different type of collagen in their bodies; it is less robust and mature than is the collagen  
9 in non-hypermobile people, and therefore people with hypermobile joints heal more slowly  
10 from injuries than do normal people.  
11

12 Dr. Graham examined Ms. Peterson on referral from her treating rheumatologist, Paul  
13 Brown, M.D. Dr. Graham testified that, because Ms. Peterson was hypermobile before this  
14 collision, she was at greater risk of injury, and once injured, at greater risk to develop chronic  
15 pain. Dr. Graham refers to this syndrome as "hypermobility syndrome" or "hypermobility  
16 joint syndrome."<sup>2</sup> Dr. Graham opined that there is a sub-group of rheumatologists who attend  
17 the American College of Rheumatology's conferences and who regularly meet to discuss  
18 hypermobility joint syndrome issues. Among that group, hypermobility joint syndrome is a  
19 diagnosis that is generally accepted, and it is generally accepted that hypermobility joint  
20  
21  
22  
23

24 <sup>2</sup> Dr. Graham and Dr. Holman both testified that "fibromyalgia" is not a recognized diagnosis outside the United  
25 States and Canada. British and European doctors simply refer to those who have chronic widespread pain as  
26 having chronic widespread pain. They do not give the syndrome a separate name.

1 syndrome can be caused by trauma such as a motor vehicle collision.<sup>3</sup> It also is generally  
2 accepted that hypermobility joint syndrome can cause chronic widespread pain, although it  
3 does not do so in everyone with the syndrome.

4 Dr. Graham testified that it is biologically plausible that trauma can cause hyper-  
5 mobility joint syndrome, because hypermobile persons have weaker collagen, are more suscep-  
6 tible to injury, and heal more slowly and/or less completely after injury than do persons who  
7 are not hypermobile. In support of his testimony, Dr. Graham noted that a number of studies  
8 have found that hypermobile athletes in various sports suffered serious injuries at a much  
9 higher rate than did their non-hypermobile teammates.

11 Plaintiff also submitted the preservation deposition of Robert Bennett, M.D. Dr. Ben-  
12 nett is board certified in rheumatology, and holds the title of "Master" in the American College  
13 of Rheumatology – a position that is reserved for physicians who are over the age of 65 and  
14 who have made outstanding contributions to the field of rheumatology. Dr. Bennett is a  
15 member of the International Myofascial Pain Society, where he served as President from 2001  
16 to 2004, and also as chairman of the board until 2007. He also has served as the program chair  
17 for the International Congress on myofascial pain and fibromyalgia, as the President of the  
18 Western Region for the American College of Rheumatology, and as an editor and a peer  
19 reviewer for numerous prestigious medical journals, including *Arthritis & Rheumatism*, *The*  
20 *Journal of the American Medical Association*, *The New England Journal of Medicine*, *Pain*,  
21 *the Journal of Musculoskeletal Pain*, *Geriatrics*, *The Journal of Functional Syndromes*, *The*  
22

24  
25 <sup>3</sup> Dr. Graham acknowledged that there may be causes of hypermobility joint syndrome other than trauma, but  
26 opined that trauma is a cause of the chronic widespread pain of hypermobility joint syndrome.

1 *Journal of Rheumatology*, and *The Journal of Clinical Investigation*. Dr. Bennett was on the  
2 American College of Rheumatology board which determined what the diagnostic criteria  
3 would be for fibromyalgia, and he personally has treated more than 10,000 fibromyalgia  
4 patients.

5 Dr. Bennett testified that it has long been well and generally accepted in medicine that  
6 assessment of the cause of a patient's illness or condition is done via a three-part epidemiologi-  
7 cal process that assesses temporality (how soon a condition arises after exposure to a suspected  
8 causal event), biologic plausibility (whether a causal relationship makes sense in light of what  
9 is known about biology, anatomy and physiology), and lack of more likely alternative causes.  
10 Dr. Bennett testified that the role of trauma as a cause of fibromyalgia in some susceptible  
11 people is generally accepted among rheumatologists who specialize in treatment of chronic  
12 pain and fibromyalgia, and by those who do research into its causes. Dr. Bennett opined that  
13 he talks to colleagues about fibromyalgia and its causes "all the time," most recently at the  
14 October 2008 American College of Rheumatology conference, where he delivered one of the  
15 opening addresses. Dr. Bennett stated: "It is my impression – I can't think of one of my  
16 immediate number of colleagues who think otherwise than that trauma is involved in the onset  
17 of fibromyalgia." Dep. of Robert Bennett at 25. Likewise, Dr. Bennett testified that there is no  
18 significant dispute in the community of physicians involved in the diagnosis, treatment,  
19 evaluation and research of fibromyalgia, that physical trauma can cause fibromyalgia to  
20 develop in some people. Consistently with plaintiff's other experts, Dr. Bennett opined that  
21 patients with fibromyalgia process pain differently from normal people, and that fibromyalgia  
22 is a condition of sensitization to painful stimuli.  
23  
24  
25  
26

1 Plaintiff also presented testimony from her treating rheumatologist, Paul Brown, M.D.  
2 Dr. Brown is a clinical professor at the University of Washington medical school, is generally  
3 familiar with the medical literature on fibromyalgia, and has treated over 3000 patients who  
4 were diagnosed with fibromyalgia. Dr. Brown's testimony was consistent with that of  
5 plaintiff's other experts. He opined that, based on his clinical experience, his review of the  
6 medical literature, his attendance at rheumatology conferences and his discussions with  
7 colleagues, it is generally accepted in the community of rheumatologists who specialize in  
8 treatment of patients with chronic pain disorders that fibromyalgia is a chronic pain syndrome  
9 with "central sensitization," and that trauma can cause fibromyalgia to develop in some people.  
10 Dr. Brown specifically disputed the defendant's contention that the relationship between  
11 trauma and fibromyalgia is controversial among rheumatologists who customarily treat,  
12 evaluate, diagnose and do research on fibromyalgia.  
13

14  
15 By contrast, none of the expert witnesses called by defendant to testify in support of the  
16 defendant's *Frye* motion specializes in the treatment of fibromyalgia or does research on fibro-  
17 myalgia, chronic widespread pain, or hypermobility joint syndrome, although all of them have  
18 treated patients who have been diagnosed with fibromyalgia.<sup>4</sup>

19  
20 Significantly, **two of defendant's three experts opined that there is not any general**  
21 **consensus in the medical community that "fibromyalgia" even exists as a specific disease**  
22 **or syndrome.**<sup>5</sup> Defendant's expert witness Lawrence Murphy, M.D., a neurologist who treats

23  
24 <sup>4</sup> It is worth noting that each expert at the *Frye* hearing acknowledged that many rheumatologists do not treat patients who have fibromyalgia.

25 <sup>5</sup>As noted above, fibromyalgia is not a diagnosis that generally is made outside of the U.S. and Canada. Dr.  
26 Graham testified that he did not regard fibromyalgia as a distinct diagnostic entity, but feels that it is a subset of

1 patients who have been diagnosed with fibromyalgia, testified via deposition that fibromyalgia  
2 itself is not a diagnosis that is generally accepted in the medical community. Dep. of Law-  
3 rence Murphy, Vol II, at 26-27. Dr. Murphy himself does not use the term "fibromyalgia"  
4 when making a diagnosis, but instead uses terms such as "myofascial pain syndrome." *Id.*

5  
6 John Dixon, MD, a board-certified rheumatologist who also testified as a defense  
7 expert, has examined and treated patients who meet the diagnostic criteria for fibromyalgia, but  
8 he himself diagnoses such patients as having "chronic widespread unexplained pain." Dr. Dix-  
9 on testified that it is **highly controversial whether fibromyalgia is even a discrete medical**  
10 **disorder.** According to Dr. Dixon, fibromyalgia is not really a disease or a syndrome, because  
11 it is not defined by any objective features. Dr. Dixon further opined that the nature of fibro-  
12 myalgia is disputed within the rheumatology community. The classification criteria for fibro-  
13 myalgia were adopted in 1990 by the American College of Rheumatology, but as Dr. Dixon  
14 explained, these classification criteria may not necessarily identify a specific disease entity.  
15

16 The classification criteria for fibromyalgia are as follows:

- 17 1. chronic widespread pain, on both sides of the body, both above and below the  
18 waist, as well as long the axial skeleton; and
- 19 2. Pain resulting from 4 Kg pressure on at least 11 of 18 designated "tender  
20 points" on the body.

21 Dr. Dixon noted that the diagnosis of fibromyalgia differs greatly between individuals, and that  
22 the value of "tender points" as a diagnostic or characteristic feature of fibromyalgia has come  
23 under increasing criticism among rheumatologists in recent years.

24  
25 the broader category of chronic widespread pain and hypermobility joint syndrome, since the symptoms are  
26 essentially the same.



1 Kent Ta, M.D., another board certified rheumatologist, also testified as an expert for  
2 defendant. Dr. Ta disagreed with Dr. Dixon in some respects. Dr. Ta testified that fibromyal-  
3 gia was a distinct clinical entity, and that the presence or absence of the requisite "tender  
4 points" distinguishes patients who have fibromyalgia from those who merely have other forms  
5 of chronic widespread pain. Interestingly, Dr. Ta opined that the medical literature established  
6 a consensus that **trauma can induce a state of widespread pain in some patients**, although  
7 he noted that such widespread pain typically resolves in less than a year. However, Dr. Ta  
8 testified that it is difficult to know when a person with chronic widespread pain caused by a  
9 traumatic injury transitions into fibromyalgia, and he was unable to say when this occurred in  
10 Ms. Peterson's case.  
11

12 Dr. Ta also agreed with other witnesses that the criterion of tender points in making a  
13 diagnosis of fibromyalgia was not necessarily desirable, since people with chronic widespread  
14 pain who have the tender points are diagnosed with fibromyalgia, and those who do not have  
15 the requisite tender points carry a different diagnosis, even though there is no real evidence that  
16 their medical conditions differ significantly in any other way. Indeed, Dr. Ta acknowledged  
17 that some prominent fibromyalgia researchers have advocated for the discarding of tender  
18 points as a diagnostic criterion for fibromyalgia. Plaintiff's expert Dr. Holman agreed, stating  
19 that "fundamental concerns about defining fibromyalgia as a single entity abound" among  
20 rheumatologists at the present time, and that there is a growing lack of confidence in the  
21 original American College of Rheumatology criteria for the diagnosis of fibromyalgia..  
22

23 This court finds from the above-described testimony that fibromyalgia/chronic wides-  
24 pread pain syndrome/hypermobility joint syndrome are medical conditions whose causes are  
25  
26

1 the subject of intense research among a relatively small percentage of rheumatologists, and that  
2 only a small percentage of rheumatologists and an even smaller percentage of physicians in  
3 general have any significant experience in studying and treating patients who have fibro-  
4 myalgia, hypermobility joint syndrome, and/or chronic widespread pain. This court therefore  
5 concludes that the "relevant scientific community" for purpose of a *Frye* analysis into the  
6 causes of such conditions is that group of physicians who specialize in researching and in  
7 treating patients with such conditions.  
8

9         Given this conclusion, plaintiff Peterson has produced sufficient evidence to show that  
10 there now is general acceptance in the relevant scientific community that her condition,  
11 whether labeled fibromyalgia, chronic widespread pain, hypermobility joint syndrome, or  
12 otherwise, is one which can be proximately caused by trauma such as this motor vehicle  
13 collision. The issue of whether or to what extent this particular patient's pain was caused by  
14 this particular collision then becomes one for the trier of fact. While large, double blinded  
15 prospective epidemiological studies have not yet been done, these should not be not required  
16 for plaintiff to be able to present her damages claims to the jury in this case, where plaintiff has  
17 shown general acceptance of her causation theory in the requisite scientific community, and  
18 where the methodology for establishing causation is neither novel nor controversial.  
19

20         **B. WHAT IS THE "SCIENTIFIC THEORY" WHOSE GENERAL ACCEPTANCE IN THE**  
21         **RELEVANT SCIENTIFIC COMMUNITY MUST BE DETERMINED BY THE COURT?**

22         In *Grant v. Boccia*, the court agreed with the defendant's contention that "the proposi-  
23 tion that trauma causes fibromyalgia is not generally accepted in the relevant scientific com-  
24 munity". *Grant*, 133 Wn. App. at 178. Although the plaintiff in *Grant* argued that his experts'  
25 opinions that the plaintiff's fibromyalgia was caused by trauma from a motor vehicle collision  
26

1 were based upon accepted scientific methodologies used in determining causation, the court  
2 found that there was no "definitive acceptance" concerning the cause of fibromyalgia, and that  
3 therefore the plaintiffs' experts should not be permitted to testify concerning causation.

4 But the instant case presents an issue that was not addressed in *Grant*. In *Grant*, the  
5 plaintiff did not provide "evidence [that] their experts' methodologies to conclude trauma  
6 causes fibromyalgia were sufficiently established to have gained general acceptance." *Grant* at  
7 180 (emphasis added). Indeed, the *Grant* court did not even address what the relevant  
8 scientific community has generally accepted to be reliable methodologies for determining  
9 medical causation. Rather, the court assumed without analysis that what *Frye* required was  
10 that the theory that "trauma can cause fibromyalgia" had to be generally accepted in the  
11 relevant scientific community, rather than the methodology of how doctors determine  
12 causation.  
13

14  
15 In contrast to the facts as set forth in *Grant v. Boccia*, Ms. Peterson has presented  
16 testimony from several well-qualified experts, including epidemiology expert Michael Free-  
17 man, Ph.D. that, in the fields of medicine and epidemiology, it has long been generally accept-  
18 ed that an event can be considered to be the cause of an injury, illness, or symptom, if the  
19 causation theory satisfies three criteria:

- 20  
21 (1) The onset of the injury, illness, or symptom has a close  
22 *temporal connection* with the event; *i.e.*, that the injury, ill-  
23 ness, or symptom manifests relatively soon after the event  
24 in question.  
25  
26 (2) A causal connection between the event and the injury or  
illness in question is *biologically plausible*, meaning that

1                   there is a hypothesis or theory that would support causation  
2                   and that does not violate known medical principles.<sup>6</sup>

3                   (3) The lack of a likely alternative explanation.

4                   Plaintiff also provided expert testimony that each of these three factors was met in Ms.  
5                   Peterson's case.

6                   1) **Temporal relationship:** Here, the evidence is undisputed that Ms. Peterson  
7                   was involved in a high speed, high property damage motor vehicle collision. She suffered a  
8                   non-displaced fracture, numerous contusions, and strains or sprains of her neck and back. She  
9                   complained of pain in several parts of her body immediately after the motor vehicle collision,  
10                  and she has never had a pain-free day since the collision. Unfortunately, instead of her injuries  
11                  resolving with time, Ms. Peterson developed widespread chronic pain, and eventually was  
12                  diagnosed with fibromyalgia, which she did not suffer from before the motor vehicle collision.  
13                  Thus, the requirement of a close temporal relationship between the motor vehicle

14                  collision and Ms. Peterson's developing chronic widespread pain is satisfied.  
15                  2) **Biologic Plausibility:** Plaintiff's experts all agreed that chronic widespread pain  
16                  and fibromyalgia are disorders in the neurological perception of pain, rather than merely  
17                  injuries to muscles, tendons and ligaments. Specifically, plaintiff's experts cited ongoing re-  
18                  search on "central sensitization", wherein a person becomes hypersensitive to painful stimuli to  
19                  the point where sensations that a "normal" person would not perceive to be painful are per-  
20                  ceived by the affected individual to be painful. For example, plaintiff's experts cited literature  
21                  22                  23

24                  <sup>6</sup>For example, as Dr. Freeman explained, the theory that a motor vehicle collision had caused a symptomatic brain  
25                  tumor within a short time after the collision is not biologically plausible, since brain tumors are known to take a  
26                  very long time to grow to the point where they cause symptoms and are diagnosed.

1 in both human and animal models, showing that once this syndrome is established, individuals  
2 with chronic widespread pain are unusually sensitive to heat, cold and other relatively benign  
3 stimuli.

4 According to plaintiff's experts, injuries to muscles, ligaments, tendons and similar soft  
5 tissues can, in certain susceptible individuals, trigger the onset of chronic widespread pain.  
6 Plaintiff's experts also agreed that persons with hypermobile joints are more susceptible to soft  
7 tissue injuries from trauma, that their injuries tend to be more severe, and that they tend to heal  
8 more slowly and more poorly than persons who do not have hypermobile joints. All experts  
9 who testified at the *Frye* hearing agreed that Ms. Peterson had hypermobile joints before the  
10 motor vehicle collision, *i.e.*, her pre-existing condition made her more susceptible to injury  
11 from this collision than a "normal" person would be.  
12

13 **3) Lack of a more likely alternative explanation:** None of the expert witnesses  
14 who testified at the *Frye* hearing suggested any explanation for Ms. Peterson's chronic wide-  
15 spread pain, hypermobility joint syndrome or fibromyalgia, other than the motor vehicle  
16 collision. In particular, none of Defendants' experts, Dr. Ta, Dr. Dixon, or Dr. Murphy, was  
17 able to provide any alternative explanation for Ms. Peterson's current chronic pain condition.  
18

19 Plaintiff's evidence is sufficient to show that the **methodology** used by her experts  
20 and her treating physicians to assess the causal relationship between her motor vehicle accident  
21 and her chronic widespread pain, hypermobility joint syndrome, and/or fibromyalgia, is not  
22 "novel", and thus does not implicate *Frye*. Moreover, to the extent that *Frye* is implicated,  
23 plaintiff has shown that this methodology is generally accepted in the relevant medical  
24 community.  
25  
26

1 C. GENERAL LEGAL PRINCIPALS THAT CONTROL IN THIS CASE

2 (1) Purpose of Tort Law: The purpose of tort law is to make an injured person  
3 whole, once a plaintiff has proved negligence, injury and causation by preponderance of the  
4 evidence. "The cornerstone of tort law is the assurance of full compensation to the injured  
5 party." *Seattle-First Nat. Bank v. Shoreline Concrete Co.*, 91 Wn.2d 230, 236 (1978). It has  
6 long been established that a primary purpose for awards of "nonpunitive, pecuniary  
7 compensation to the injured party is to repair [her] injury, or to make [her] whole again as  
8 nearly as that may be done by an award of money." *DeNike v. Mowery*, 69 Wn.2d 357, 358  
9 (1966).  
10

11 (2) Proximate Cause: As in any personal injury case, Ms. Peterson is not re-  
12 quired to prove that the motor vehicle collision with the defendant was the sole cause of her  
13 chronic pain condition. She need only prove that the collision was a proximate cause of her  
14 condition. *See* WPI 15.01.  
15

16 To some extent, Ms. Peterson's causation theory was supported even by the testimony  
17 of one of the defendant's experts, Dr. Kent Ta. Dr. Ta testified that, based on the available  
18 medical literature, **trauma can induce a state of chronic widespread pain in some patients.**<sup>7</sup>  
19 He expressed the opinion that in Ms. Peterson's case, the motor vehicle collision lighted up an  
20 inherent predisposition that produced the chronic pain that Ms. Peterson suffers from today.  
21

22 Such testimony tacitly accepts the motor vehicle collision as a proximate cause of Ms.  
23 Peterson's chronic widespread pain, because a plaintiff with a preexisting, asymptomatic con-  
24

25 <sup>7</sup> While Dr. Ta added that this condition usually does not last for more than a year, his testimony appears  
26 to support plaintiff's claim that her motor vehicle collision caused her chronic widespread pain.

1 | dition that is lighted up as a result of a tortious injury is entitled to full recovery. See Washing-  
2 | ton Pattern Instruction (WPI) 30.17 and 30.18. "When an accident lights up and makes active  
3 | a preexisting condition that was dormant and asymptomatic immediately prior to the accident,  
4 | the preexisting condition is not a proximate cause of the resulting damages." *Harris v. Drake*,  
5 | 152 Wn.2d 480, 494 (2004). Nor would evidence that plaintiff had an unusual susceptibility  
6 | to this type of injury constitute a defense to her claim for damages. See WPI 30.18.01.  
7 |

8 |         (3) Burden of Proof: Plaintiff's burden of proof is by a preponderance of the  
9 | evidence. In order for a plaintiff to prove that a particular tortious event was a proximate cause  
10 | of an injury, she only must present sufficient medical testimony to allow a reasonable person to  
11 | infer that the causal connection exists. "If, from the facts and circumstances and the medical  
12 | testimony given, a reasonable person can infer that the causal connection exists, the evidence is  
13 | sufficient." *McLaughlin v. Cooke*, 112 Wn.2d 829, 837-838 (1989).  
14 |

15 |         For example, in *Vanderhoff v. Fitzgerald*, 72 Wn.2d 103, 108 (1967), the Supreme  
16 | Court held that it was proper to allow a physician to testify to his opinion that a plaintiff's  
17 | motor vehicle accident injuries accelerated the growth of her bladder cancer.

18 |         [The doctor] is a licensed physician with considerable experience in  
19 | cancer research and he displayed familiarity with the disease. He was  
20 | an attending physician. He based his opinion on history, his examina-  
21 | tion of the patient, hospital reports, reports of consulting physicians  
22 | and laboratory reports. We find no error in permitting him to express  
23 | an opinion regarding Mrs. Vanderhoff's condition and its cause.

24 |         In this case, Ms. Peterson claims that she was pain-free before the subject motor vehicle  
25 | collision, and that ever since the collision, she has suffered from severe pain. She has present-  
26 | ed testimony from numerous well-qualified expert rheumatologists with considerable exper-  
27 | ience in chronic widespread pain, fibromyalgia and hypermobility joint syndrome. They based

1 their opinions on Ms. Peterson's history, their physical examinations, hospital records, reports  
2 of consulting physicians and laboratory reports, as well as on their own research, their review  
3 of peer-reviewed medical literature, and their discussions with colleagues. As in *Vanderhoff*,  
4 these doctors should be permitted to express their opinions concerning Ms. Peterson's condi-  
5 tion and the causal connection between her chronic pain and the motor vehicle collision with  
6 the defendant. It will be for the trier of fact to determine what weight, if any, to give these  
7 opinions. Regardless of the label different doctors may place on her condition, it would be  
8 manifestly unjust not to permit Ms. Peterson to place her claims before a jury, and allow the  
9 jury to determine whether the collision was or was not a proximate cause of Ms. Peterson's  
10 widespread chronic pain.  
11

12 DATED this 12<sup>th</sup> day of February, 2009.

13  
14  
15 

16 Andrea Darvas  
17 Superior Court Judge  
18  
19  
20  
21  
22  
23  
24  
25  
26